Signaling and Communication in Plants

P. Vidhyasekaran

Switching on Plant Innate Immunity Signaling Systems

Bioengineering and Molecular Manipulation of PAMP-PIMP-PRR Signaling Complex

Springer

Signaling and Communication in Plants

Series Editors
František Baluška
Department of Plant Cell Biology, IZMB, University of Bonn, Kirschallee 1,
D-53115 Bonn, Germany

Jorge Vivanco
Center for Rhizosphere Biology, Colorado State University, 217 Shepardson Building,
Fort Collins, CO 80523-1173, USA

More information about this series at http://www.springer.com/series/8094

P. Vidhyasekaran

Switching on Plant Innate Immunity Signaling Systems

Bioengineering and Molecular Manipulation of PAMP-PIMP-PRR Signaling Complex

Springer

P. Vidhyasekaran
Plant Pathology
Tamil Nadu Agricultural university
Coimbatore, Tamil Nadu, India

ISSN 1867-9048 ISSN 1867-9056 (electronic)
Signaling and Communication in Plants
ISBN 978-3-319-26116-4 ISBN 978-3-319-26118-8 (eBook)
DOI 10.1007/978-3-319-26118-8

Library of Congress Control Number: 2016930022

Springer Cham Heidelberg New York Dordrecht London

Printed on acid-free paper

Springer International Publishing AG Switzerland is part of Springer Science+Business Media (www.springer.com)

Contents

Chapter 1
Introduction

Abstract Plant innate immune system is a surveillance system against possible attack by pathogens. It is quiescent in normal healthy plants. It is a sleeping giant and when awakened by specific signals it triggers expression of several defense genes. Unlike, transgenic plants developed by engineering disease resistance genes against specific pathogens, plants overexpressing the plant immune system awakened by the alarm signals PAMP and PIMP trigger expression of hundreds of defense genes conferring resistance against wide range of pathogens. Both PAMPs and PIMPs are perceived by plants as alarm signals by specific receptors called pattern recognition receptors (PRRs). PAMPs activate expression of the genes encoding various PRRs. Besides PAMP molecules, pathogens secrete another type of molecules called effectors. While the pathogen-derived PAMPs are involved in switching-on the plant immune responses, the effectors are involved in switching-off the PAMP-triggered innate immunity. The effectors may also bind with PRRs and disrupt binding of PAMP with PRR in PAMP-PRR signaling complex to impede PAMP-triggered plant immunity. Effectors may bind with the PRR signal amplifier BAK1 and block the function of PAMP-PRR signaling complex. Early and robust activation of PAMP-PRR signaling complex before the pathogens invade and secrete virulence effectors seems to be necessary for triggering strong defense responses. Several PAMP formulations have been developed and foliar application of the formulations triggers the induction of plant immune responses. The time of application is very critical in enhancing the efficacy of the PAMPs in controlling diseases. The PAMPs should be applied prior to pathogen invasion. The concentration of the PAMP applied also determines the efficacy of the treatment in controlling diseases. Oligogalacturonates (OGAs), plant elicitor peptides (Peps), and PAMP-induced Peptides (PIPs) are the important PIMPs capable of switching on plant innate immune responses. Bioengineering technologies have been exploited to utilize PIMPs to develop transgenic plants expressing enhanced disease resistance. Bioengineering PRRs has been shown to be another potential technology to awaken the quiescent plant innate immunity for effective management of crop diseases. Intergeneric transfer of PRR has been achieved to develop disease-resistant crop plants. Transcription factors are the master switches, which regulate expression of defense genes in the PAMP-triggered plant immune signaling systems. Several transcription factors have been shown to trigger "priming" of defense responses and induce "Systemic Acquired resistance (SAR)" in plants. The plant defense activators benzothiadiazole, probenazole, tiadinil and ergosterol trigger the expression of

P. Vidhyasekaran, *Switching on Plant Innate Immunity Signaling Systems*,
Signaling and Communication in Plants, DOI 10.1007/978-3-319-26118-8_1

transcription factors which modulate the expression of defense genes inducing disease resistance. The rhizobacterial strain *Pseudomonas fluorescens* WCS417r induces systemic resistance (ISR) by activating the transcription factor MYB72. The MYC2 transcription factor also has been shown to be involved in *P. fluorescens* WCS417r-induced priming to trigger ISR. Several WRKY, MYB, MYC, bZIP, EREBP, and NAC transcription factors have been engineered in various crop plants to develop disease resistant plants. Some transcription factors have been found to negatively regulate the expression of defense signaling systems. Silencing of the negative regulator transcription factors may be a useful strategy in developing disease-resistant plants. Several bioengineering and molecular manipulation technologies have been developed to switch on the 'sleeping' plant innate immune system, which has potential to detect and suppress the development of a wide range of plant pathogens in economically important crop plants. Enhancing disease resistance through altered regulation of plant immunity signaling systems would be durable and publicly acceptable.

1.1 Plant Innate Immunity Is a Sleeping Giant to Fight against Pathogens

Diseases caused by oomycete, fungal, bacterial, and viral pathogens cause enormous crop losses and in some areas the crop may be completely devastated (Vidhyasekaran 2004; Byamukama et al. 2015; Cohen et al. 2015; Han et al. 2015; Handiseni et al. 2015; Holmes et al. 2015; Sharma-Poudyal et al. 2015; Strehlow et al. 2015). Chemical control is widely practiced to manage fungal and oomycete pathogens (Gent et al. 2015; Handiseni et al. 2015). Frequent development of resistance to the modern fungicides in the field population of fungal/oomycete pathogens is a challenging problem in using the fungicides to manage these diseases (Miles et al. 2012; Gudmestad et al. 2013; Tymon and Johnson 2014; Fernández-Ortuño et al. 2015; Hu et al. 2015; Keinath 2015; Saville et al. 2015; Zeng et al. 2015). Effective chemicals are still not available to control bacterial, viral, viroid, and phytoplasma diseases (Jones 2001; Vidhyasekaran 2004; Bradley 2008; Kanetis et al. 2008). Breeding varieties with built-in resistance may be the sound approach to manage diseases (Vidhyasekaran 2007; Singh et al. 2008; Tagle et al. 2015). However, new races of pathogens appear frequently and the resistance often breaks down (Sørensen et al. 2014; Kitner et al. 2015; Maccaferri et al. 2015). Breeding for quantitative resistance is useful (Yasuda et al. 2015), but it is difficult to achieve (Vidhyasekaran 2007; St Clair 2010; Zhang et al. 2015). Breeding for resistance against broad-spectrum of pathogens will be ideal, but the traditional breeding methods are inefficient (Vleeshouwers et al. 2008; Tran et al. 2015). An alternative technology based on switching on plant innate immunity using pathogen-associated molecular patterns (PAMPs) and pathogen-induced molecular patterns (PIMP)/host-associated molecular patterns (HAMPs) has been developed recently for

management of viral and bacterial diseases (Li et al. 2011; Pavli et al. 2011, 2012; Choi et al. 2012; Li et al. 2012), and also for management of a wide range of biotrophic, hemibiotrophic, and necrotrophic fungal and oomycete pathogens (Miao et al. 2010; Xu et al. 2010; Choi et al. 2012; Miao and Wang 2013).

Plant innate immune system is a surveillance system against possible attack by pathogens. It is quiescent in normal healthy plants. It is a sleeping giant and when awakened by specific signals it triggers expression of several defense genes. Several hundreds of defense genes have been detected in plants and these are involved in plant immunity (Coram and Pang 2005; Vega-Sánchez et al. 2005; Wang et al. 2005; Wilkinson et al. 2005; Hermosa et al. 2006; Vidhyasekaran 2007; Sun et al. 2008). These defense genes encode different pathogenesis-related proteins (Anand et al. 2003; Zhu et al. 2006; Sun et al. 2008) most of which inhibit growth of oomycete (Lee et al. 2000), fungal (Moravčikova et al. 2004; Pervieux et al. 2004; Chen et al. 2006; Zhu et al. 2006; Hernández-Blanco et al. 2007) and bacterial pathogens (Vidhyasekaran 2002). Several defense-related genes encode enzymes involved in biosynthesis of toxic compounds such as phytoalexins (Nawar and Kuti 2003; Liu et al. 2006; Chassot et al. 2008), phenylpropanoids and isoflavonoids (Farag et al. 2008), and terpenoids (Keeling et al. 2008), or enzymes involved in cell wall-fortification (Hamiduzzaman et al. 2005; Flors et al. 2008). The triggered plant immune responses include accumulation of pathogenesis-related proteins, deposition of lignin and callose in the cell wall, and production of anti-microbial compounds (Tsuda and Katagiri 2010; Gimenez-Ibanez and Rathjen 2010). Plant innate immunity is a powerful weapon to fight against a wide range of plant pathogens. Plants have innate immunity system (Nicaise 2014; Vidhyasekaran 2014, 2015; Piasecka et al. 2015; Robinson and Bostock 2015; Schwessinger et al. 2015; Tena 2015) and this system provides basic protective functions against broadest range of pathogens (Boller and He 2009; Boutrot et al. 2010; Chen et al. 2010a, b; Dodds and Rathjen 2010; Park et al. 2010b; Shimizu et al. 2010; Segonzac and Zipfel 2011; Zamioudis and Peterse 2012; Li et al. 2014a, b; Vidhyasekaran 2014).

1.2 Potential Signals to Switch on Plant Immune System

The plant innate immune systems have high potential to fight against viral, bacterial, oomycete, and fungal pathogens and protect the crop plants against wide range of diseases (Knecht et al. 2010; Lacombe et al. 2010; D'Amelio et al. 2011; Hwang and Hwang 2011; Alkan et al. 2012). However, these plant immune systems are quiescent in healthy normal plants. Specific signals are needed to switch on the sleeping giant for exploiting the quiescent immune system for combating diseases. These signals are derived from invading pathogens and called 'pathogen-associated molecular patterns' (PAMPs). The immune system is activated on perception of the PAMP of invading pathogens (Nürnberger and Kufner 2011; Segonzac and Zipfel 2011). Potential pathogens contain several PAMPs and they serve as alarm signals to activate the plant innate immunity (Böhm et al. 2014; Vidhyasekaran 2014;

Zhang et al. 2014). PAMPs are detected not only in pathogens, but also in saprophytes, probably in all microbes. Hence the PAMPs are also called as microbe-associated molecular patterns (MAMPs) (Jeworutzki et al. 2010; Thomma et al. 2011). PAMPs/MAMPS are potential tools to activate plant immune systems and can be effectively used to manage crop diseases (Iriti et al. 2011; Choi et al. 2012; Dafermos et al. 2012; Li et al. 2012; Sanchez et al. 2012; Quang et al. 2015; Sathiyabama et al. 2014).

Besides the microbe-derived elicitors (MAMPs), some host plant-derived elicitors called pathogen-induced molecular patterns (PIMPs) or host-associated molecular patterns (HAMPs) have been shown to activate the plant innate immune system (Yamaguchi and Huffaker 2011; Vallarino and Osorio 2012; Bellincampi et al. 2014; Hou et al. 2014). The PIMPs/HAMPs (host-derived elicitors) function almost in the same fashion as the PAMPs function in switching on plant innate immunity (Denoux et al. 2008; Ferrari et al. 2013). PIMPs and PAMPs activate similar downstream responses using many of the same molecular components (Ryan et al. 2007; Krol et al. 2010; Postel et al. 2010; Qi et al. 2010; Yamaguchi et al. 2010; Huffaker et al. 2011). Both of them bind specific LRR receptors and both activate the same downstream signaling events (Yamaguchi et al. 2006; Huffaker and Ryan 2007; Krol et al. 2010).

Both PAMPs and PIMPs are perceived by plants as alarm signals by specific receptors called pattern recognition receptors (PRRs) (Nicaise et al. 2009; Brutus et al. 2010; Petutschnig et al. 2010; Shinya et al. 2010; Schulze et al. 2010; Segonzac and Zipfel 2011; Hann et al. 2014). Plants utilize the PRRs to recognize PAMPs/MAMPs (Böhm et al. 2014; Macho and Zipfel 2014; Zhang et al. 2014). Most of the PRRs identified are receptor-like kinases (RLKs) and receptor-like proteins (RLPs) (Yang et al. 2012; Zhang et al. 2013; Zipfel 2014). PAMPs activate expression of the genes encoding various PRRs (Zipfel et al. 2004, 2006; Qutob et al. 2006; Lohmann et al. 2010). PRRs bind with PAMPs for their activation (Boutrot et al. 2010; Petutschnig et al. 2010). The PRRs recognize PAMPs and PIMPs and switch on the plant innate immunity (Mentlak et al. 2012).

1.3 Pathogens Possess Weapons to Switch-Off Plant Immune Systems

Besides PAMP molecules, pathogens secrete another type of molecules called effectors (Wu et al. 2011; Vleeshouwers and Oliver 2014). While the pathogen-derived PAMPs are involved in switching-on the plant immune responses, the effectors are involved in switching-off the PAMP-triggered innate immunity (Thomma et al. 2011; Wu et al. 2011; Cheng et al. 2012). The effectors secreted by various pathogens have been shown to suppress the PAMP-triggered immunity (Shan et al. 2008; de Jonge et al. 2010). Effectors induce susceptibility, mostly by suppressing PAMP-induced immune responses. The effector proteins target basic innate immunity in

plants (Boller and He 2009; Song and Yang 2010; Szczesny et al. 2010; Rajput et al. 2014; Zheng et al. 2014). Several bacterial pathogens use a specialized type III secretion system to deliver effector proteins into host cells to subvert PAMP-triggered host defense mechanisms, thereby promoting pathogenesis (Song and Yang 2010; Szczesny et al. 2010; Zhang et al. 2010; Wu et al. 2011; Akimoto-Tomiyama et al. 2012).

The effectors may also bind with PRRs and block plant defense responses in the plant cell (Xiang et al. 2008; Zeng et al. 2012; Rosli et al. 2013; Xin and He 2013). Effectors may disrupt binding of PAMP with PRR in PAMP-PRR signaling complex to impede PAMP-triggered plant immunity (Mentlak et al. 2012). Some effectors have been shown to degrade the PRRs through ubiquitin-proteasome pathway and inhibit PAMP-triggered immunity (Göhre et al. 2008; Gimenez-Ibanez et al. 2009a, b). Effectors may also target the kinase domains of PRR and inhibit the PRR receptor kinase activity to block PAMP-triggered immunity (Shan et al. 2008; Xiang et al. 2008; Zipfel and Rathjen 2008; Xiang et al. 2011). The effectors may also inhibit the autophosphorylation of PRRs to suppress the PAMP-triggered immune system (Xiang et al. 2008). The effectors may prevent the activation of PRR signaling complex by inhibiting the autophosphorylation of PRRs (Hann and Rathjen 2007). Effectors may bind with the PRR signal amplifier BAK1 and block the function of PAMP-PRR signaling complex (Shan et al. 2008; Xiang et al. 2008; Hann et al. 2010).

1.4 Bioengineering and Molecular Manipulation Technologies to Switch on the Sleeping Quiescent Plant Immune System to Win the War against Pathogens

Several bioengineering and molecular manipulation technologies have been developed for management of a broad-spectrum of diseases caused by a wide-range of viral, bacterial, fungal and oomycete pathogens, exploiting the potential of plant innate immunity (Ferrari et al. 2008; Lacombe et al. 2010; Hwang and Hwang 2011; Volpi et al. 2011; Alkan et al. 2012; Ferrari et al. 2012; Li et al. 2012; Wang et al. 2013; Fu et al. 2014; Lloyd et al. 2014; Macho and Zipfel 2014; Trouvelot et al. 2014). The crop diseases can be controlled by switching on plant innate immunity by manipulating PAMP-PIMP-PRR signaling complex. Early, rapid and strong activation of plant innate immune system is necessary to induce strong defense responses against pathogens. Early and robust activation of PAMP-PRR signaling complex before the pathogens invade and secrete virulence effectors seems to be necessary for triggering strong defense responses and for effective management of crop diseases (Orlowska et al. 2011; Aghnoum and Niks 2012; Lanubile et al. 2012).

Several PAMP formulations have been developed and foliar application of the formulations triggered the induction of plant immune responses (Dong et al. 2004;

Elmer and Reglinski 2006; de Capdeville et al. 2008; Shao et al. 2008; Iriti et al. 2011; Dafermos et al. 2012; Chuang et al. 2014). Several factors such as environment, genotype, and crop nutrition determine the efficacy of the PAMPs in controlling diseases under field conditions (Walters et al. 2005). The time of application is very critical in enhancing the efficacy of the PAMPs in controlling diseases (de Capdeville et al. 2002; Agostini et al. 2003). The PAMPs should be applied 2–5 days prior to pathogen invasion (Qiu et al. 2001; de Capdeville et al. 2002, 2003). The concentration of the PAMP applied also determines the efficacy of the treatment in controlling diseases (de Capdeville et al. 2002; Chen et al. 2008). Variability in structure and function has been reported among various PAMPs (Che et al. 2000; Tanaka et al. 2003; Fujiwara et al. 2004). The time of induction (Luna et al. 2011), intensity of induction (Lecourieux et al. 2002, 2005), and duration of induction (Aziz et al. 2007) of the defense signals may vary depending on the type of PAMPs. Efficacy of PAMPs in controlling diseases may also vary depending on the challenging pathogens (Agostini et al. 2003). Hence, suitable PAMPs have to be selected for management of various crop diseases.

Bioengineering PAMP genes has been shown to be powerful tool to trigger plant immune responses (Keller et al. 1999; Li and Fan 1999; Belbahri et al. 2001; Choi et al. 2004; Donghua et al. 2004; Peng et al. 2004; Takakura et al. 2004, 2008; Malnoy et al. 2005; Jang et al. 2006; Ren et al. 2006a, b; Cai et al. 2007; Sohn et al. 2007; Shao et al. 2008; Qiu et al. 2009; Huo et al. 2010; Miao et al. 2010; Xu et al. 2010; Pavli et al. 2011, 2012; Choi et al. 2012; Li et al. 2012; Miao and Wang 2013; Quang et al. 2015). Levels of PAMP gene expression may vary among different transgenic plant lines developed by bioengineering technologies (Peng et al. 2004). The line, which shows high level of PAMP gene expression, shows very high level of resistance against pathogens, while the line, which shows low level of expression of the PAMP gene shows only low level of resistance (Peng et al. 2004). Hence, the transgenic lines should be carefully selected to generate highly useful disease-resistant cultivars. Expression of PAMP genes can be enhanced by properly selecting the promoter for gene transcription (Takakura et al. 2004). The transgenic plants expressing introduced gene may have side effects, showing retardation of plant growth and reduced crop yield potential. However, transgenic plants expressing the PAMP harpin gene show good agronomic characters (Xu et al. 2010; Li et al. 2011; Pavli et al. 2011). Selection of suitable pathogen-inducible promoter for expressing the PAMP gene appears to be a perquisite for developing disease-resistant plants without any reduction in yield potential (Choi et al. 2004; Donghua et al. 2004)

Bioengineering technologies have been exploited to utilize PIMPs/HAMPs to develop transgenic plants expressing enhanced disease resistance. Oligogalacturonides are the best-characterized plant cell wall-derived PIMPs/HAMPs (Vallarino and Osorio 2012). However, not all OGAs are capable of eliciting a defense response. Their ability to elicit defense responses depends on length (degree of polymerization), degree of methyl esterification and the level of acetylation (Côté and Hahn 1994; Vidhyasekaran 1997, 2007; Wiethölter et al. 2003; Aziz et al. 2004; Ferrari et al. 2007; Osorio et al. 2008; Vallarino and Osorio 2012).

Both the degrees of substitution (methylesterification and/or acetylation) and polymerization can be controlled by specific enzymes such as pectin methylesterases (PMEs), pectin acetylesterases (PAEs), polygalacturonases (PGs), or pectate lyases-like (PLLs) (Sĕnĕchal et al. 2014). PME can modify the structure of OGAs and the modified OGAs will be highly active in triggering plant innate immune signaling systems. Transgenic plants expressing pectin methyl esterase gene (*PME*) generate oligogalacturonides, which act as host-derived elicitor/PIMP/HAMP. These transgenic plants show enhanced expression of plant immune responses and enhanced disease resistance (Lionetti et al. 2007, 2014; Osorio et al. 2008, 2011). PME activity is tightly regulated by an inhibitor protein called pectin methylesterase inhibitor protein (PMEI) (Giovane et al. 2004; Di Matteo et al. 2005). Transgenic plants overexpressing genes encoding PME inhibitor proteins show enhanced disease resistance (Lionetti et al. 2007, 2014; An et al 2008).

Transgenic plants expressing PG gene show enhanced disease resistance (Ferrari et al. 2008). Polygalacturonase-inhibiting proteins (PGIPs) play important role in switching on plant immune signaling systems (Manfredini et al. 2005; Federici et al. 2006; Alexandersson et al. 2011). Transgenic plants expressing *PGIP* genes also show enhanced disease resistance (Joubert et al. 2006, 2007; Alexandersson et al. 2011; Borras-Hidalgo et al. 2012; Nguema-Ona et al. 2013; Wang et al. 2013). The expression of *PGIP* genes does not affect the agronomic characters of the transformed plants (Powell et al. 2000; Capodicasa et al. 2004; Agüero et al. 2005; Borras-Hidalgo et al. 2012; Nguema-Ona et al. 2013).

Plant elicitor peptides (Peps) are the other group of PIMPs/HAMPs (Huffaker et al. 2011; Logemann et al. 2013; Hann et al. 2014). The Pep proteins are processed from the precursor PROPEP proteins. Transgenic plants overexpressing *PROPEP* genes show enhanced disease resistance (Huffaker et al. 2006). Transgenic plants overexpressing PIP (PAMP-induced Peptides) and systemin switch on plant innate immunity and show enhanced disease resistance (Coppola et al. 2014; Hou et al. 2014). PIMPs/HAMPs appear to be powerful tools to engineer disease resistance in field crops.

Bioengineering PRRs has been shown to be another potential technology to awaken the quiescent plant innate immunity for effective management of crop diseases. Intergeneric transfer of PRR from the weed plant Arabidopsis to various crop species has been achieved to develop disease-resistant crop plants. EFR is a Brassicaceae-specific PRR (Zipfel et al. 2006). Transfer of EFR from *Arabidopsis* to various crop plants is highly useful for crop disease management. Pathogens that are adapted to a particular host plant may be adept at suppressing the PRRs of that host by their effectors. The effectors of the pathogens might not recognize PRRs from other host plants and development of transgenic plants expressing PRRs from other plant species may provide good resistance against various bacterial pathogens possessing the PAMP EF-Tu (Lacombe et al. 2010). Transgenic tomato plants expressing *EFR* gene from *Arabidopsis* show enhanced resistance against the tomato wilt pathogen *Ralstonia solanacearum* (Lacombe et al. 2010). Transgenic tobacco expressing EFR also show resistance against *Agrobacterium. tumefaciens* (Brutus et al. 2010). Transgenic banana plants expressing the rice PRR *XA21* gene

showed complete resistance to *Xanthomonas campestris* pv. *musacearum* (Tripathi et al. 2014). Transgenic *Citrus sinensis* plants expressing the rice PRR *XA21* gene enhance resistance against the citrus canker pathogen *Xanthomonas axonopodis* pv. *citri* (Mendes et al. 2010). Transgenic rice plants overexpressing *Xa21* gene showed enhanced resistance against the rice bacterial blight pathogen *Xanthomonas oryzae* pv. *oryzae* (Park et al. 2008, 2010a, b; Chen et al. 2014). Transgenic *Arabidopsis* plants expressing *FLS2* gene showed increased resistance against *Pseudomonas syringae* pv. *tomato* DC3000 (De Lorenzo et al. 2011). The transgenic rice plants overexpressing the HAMP receptor *WAK1* show enhanced disease resistance (Li et al. 2009).

Transcription factors are the master switches, which regulate expression of defense genes in the PAMP-triggered plant immune signaling systems (Century et al. 2008; Moreau et al. 2012). PAMPs and PIMPs/HAMPs switch on the expression of various transcription factor genes involved in plant defense responses (Denoux et al. 2008; Higashi et al. 2008; Chujo et al. 2013; McLellan et al. 2013). Several transcription factors have been shown to trigger "priming" of defense responses and induce "Systemic Acquired resistance (SAR)" in plants (Chavan and Kamble 2013; Nakayama et al. 2013). The plant defense activators benzothiadiazole, probenazole, and tiadinil trigger the expression of transcription factors which modulate the expression of defense genes inducing disease resistance (Shimono et al. 2007, 2012). DL-3-aminobutyric acid (β-aminobutyric acid, BABA) has been found to induce priming of WRKY transcription factors and trigger systemic resistance (Jakab et al. 2001). Foliar spray with BABA led to a significant reduction of lesion development in *Brassica carinata* caused by *Alternaria brassicae* (Chavan and Kamble 2013). Ergosterol treatment triggered a 23-fold increase of *VvWRKY* gene expression in grape plantlets and induced resistance against the necrotrophic fungal pathogen *Botrytis cinerea* (Laquitaine et al. 2006). The rhizobacterial strain *Pseudomonas fluorescens* WCS417r induces systemic resistance (ISR) in *A. thaliana* by activating the transcription factor MYB72 (Van der Ent et al. 2008). The MYC2 transcription factor also has been shown to be involved in *P. fluorescens* WCS417r-induced priming to trigger ISR (Pozo et al. 2008).

Several WRKY, MYB, MYC, bZIP, EREBP, and NAC transcription factors, have been engineered in various crop plants to develop disease resistant plants (He et al. 2001; Shin et al. 2002; Fischer and Dröge-Laser 2004; Guo et al. 2004; Cao et al. 2006; Waller et al. 2006; Chujo et al. 2007; Kim et al. 2007; Marchive et al. 2007; Mzid et al. 2007; Qiu et al. 2007; Wang et al. 2007; Zuo et al. 2007; Dai et al. 2008; Zhang et al. 2008; Bahrini et al. 2011a, b; Fan et al. 2011; Abbruscato et al. 2012; Liu et al. 2012; Peng et al. 2012; Shimono et al. 2012; Yu et al. 2012; Zhu et al. 2012; Han et al. 2013; Lee et al. 2013; Marchive et al. 2013; Wei et al. 2013; Chujo et al. 2014; Dang et al. 2014; Yokotani et al. 2014; Cao et al. 2016; Cheng et al. 2015; Jisha et al. 2015; Li et al. 2015; Shan et al. 2015).

Most of the successful stories in management of crop diseases using transcription factors are in rice plants. Transgenic rice plants overexpressing *OsWRKY13* showed enhanced blast (*Magnaporthe oryzae*) and bacterial blight (*Xanthomonas oryzae* pv. *oryzae*) disease resistance (Qiu et al. 2007; Cheng et al. 2015). WRKY30-

overexpressing rice plants show resistance against the rice blast pathogen *M. oryzae*, the sheath blight pathogen *Rhizoctonia solani* and the bacterial blight pathogen *X. oryzae* pv. *oryzae* (Peng et al. 2012; Han et al. 2013; Lee et al. 2013) Transgenic rice plants overexpressing *OsWRKY31* (Zhang et al. 2008), *WRKY45* (Shimono et al. 2007, 2012; Goto et al. 2015), *OsWRKY47* (Wei et al. 2013), *WRKY53* (Chujo et al. 2007, 2014), OsWRKY89 (Wei et al. 2013) show enhanced resistance to *M. oryzae.* Transgenic rice plants overexpressing *OsWRKY71* showed enhanced resistance to the bacterial blight pathogen *Xanthomonas oryzae* pv. *oryzae* (Liu et al. 2007).

Some transcription factors have been found to negatively regulate the expression of defense signaling systems. Silencing of the negative regulator transcription factors may be a useful strategy in developing disease-resistant plants. The *TaNAC1* gene-silenced wheat plants showed enhanced resistance against the stripe rust pathogen (Wang et al. 2015). *WRKY42*-suppressing (*WRKY42*-RNA interference [RNAi]) rice plants were developed and these plants showed increased resistance to *M. oryzae* (Cheng et al. 2015).

1.5 Switching on Plant Innate Immunity Using PAMP-PIMP-PRR-Transcription Factor Is the Most Potential Biotechnological Approach for Management of Crop Diseases

Susceptibility and resistance are two sides of the same coin (Vidhyasekaran 2007). The plant immune system is induced faster and to a higher level in resistant interactions (Makandar et al. 2006). The major differences between susceptible and resistant interactions are the magnitude and timing of induction of plant immune signaling system (Makandar et al. 2006; Rinaldi et al. 2007; Asselbergh et al. 2008). Higher and faster expression of genes involved in signal transduction systems has been found to be associated with improved tolerance to pathogens (Coppinger et al. 2004; Waller et al. 2006; Yamamizo et al. 2006; Zhang et al. 2006; Brader et al. 2007; Qiu et al. 2007). PAMP-induced defense in susceptible host plants is insufficient to stop infection; nonetheless, it is referred to as basal resistance (Nürnberger and Lipka 2005; Fung et al. 2008). Early, rapid and strong activation of plant innate immune system is necessary to induce strong defense responses against pathogens (Orlowska et al. 2011; Aghnoum and Niks 2012; Lanubile et al. 2010, 2012, 2014; Groβkinsky et al. 2012). Strong signals are needed to switch on early and strong activation of plant immunity. Engineering and/or proper application of PAMP/PRR products much before pathogen invasion results in early switching on the plant immune system.

Recently several bioengineering and molecular manipulation technologies have been developed to switch on the ‘sleeping’ plant innate immune system, which has potential to detect and suppress the development of a wide range of plant pathogens

in economically important crop plants (Lacombe et al. 2010). Enhancing disease resistance through altered regulation of plant immunity signaling systems would be durable and publicly acceptable (Yamamizo et al. 2006; Shao et al. 2008; Gust et al. 2010; Lacombe et al. 2010). The plant innate immune systems have high potential to fight against viral, bacterial, oomycete, and fungal pathogens and protect the crop plants against wide range of diseases (Knecht et al. 2010; Lacombe et al. 2010; D'Amelio et al. 2011; Hwang and Hwang 2011; Alkan et al. 2012). This book describes various bioengineering and molecular manipulation technologies employed to trigger defense responses and manage crop diseases.

References

Abbruscato P, Nepusz T, Mizzi L, Del Corvo M, Morandini P, Fumasoni I, Michel C, Paccanaro A, Guiderdoni E, Schaffrath U, Morel J-B, Piffanelli P, Faivre-Rampant O (2012) *OsWRKY22*, a monocot *WRKY* gene, plays a role in the resistance response to blast. Mol Plant Pathol 13:828–841

Aghnoum R, Niks RE (2012) Compatible *Puccinia hordei* infection in barley induces basal defense to subsequent infection by *Blumeria graminis*. Physiol Mol Plant Pathol 77:17–22

Agostini JP, Bushong PM, Timmer LW (2003) Greenhouse evaluation of products that induce host resistance for control of scab, melanose, and Alternaria brownspot of citrus. Plant Dis 87:69–74

Agüero CB, Uratsu SL, Greve C, Powell ALT, Labavitch JM, Meredith CP, Dandekar AM (2005) Evaluation of tolerance to Pierce's disease and *Botrytis* in transgenic plants of *Vitis vinifera* L. expressing the pear PGIP gene. Mol Plant Pathol 6:43–51

Akimoto-Tomiyama C, Furutani A, Tsuge S, Washington EJ, Nishizawa Y, Minami E, Ochiai H (2012) XopR, a type III effector secreted by *Xanthomonas oryzae* pv. *oryzae*, suppresses microbe-associated molecular pattern-triggered immunity in *Arabidopsis thaliana*. Mol Plant Microbe Interact 25:505–514

Alexandersson E, Becker JVW, Jacobson D, Nguema-Ona E, Steyn C, Denby KJ, Vivier MA (2011) Constitutive expression of a grapevine polygalacturonase-inhibiting protein affects gene expression and cell wall properties in uninfected tobacco. BMC Res Notes 4:493

Alkan N, Fluhr R, Prusky D (2012) Ammonium secretion during *Colletotrichum coccodes* infection modulates salicylic and jasmonic acid pathways of ripe and unripe tomato fruit. Mol Plant Microbe Interact 25:85–96

An SH, Sohn KH, Choi HW, Hwang IS, Lee SC, Hwang BK (2008) Pepper pectin methylesterase inhibitor protein CaPMEI1 is required for antifungal activity, basal resistance and abiotic stress tolerance. Planta 228:61–78

Anand A, Schmelz EA, Muthukrishnan S (2003) Development of a lesion-mimic phenotype in a transgenic wheat line over expressing genes for pathogenesis-related (PR) proteins is dependent on salicylic acid concentration. Mol Plant Microbe Interact 16:916–925

Asselbergh B, De Vleesschauwer D, Höfte M (2008) Global switches and fine-tuning ABA modulates plant pathogen defense. Mol Plant Microbe Interact 21:709–719

Aziz A, Heyraud A, Lambert B (2004) Oligogalacturonide signal transduction, induction of defence-related responses and protection of grapevine against *Botrytis cinerea*. Planta 218:767–774

Aziz A, Gauthier A, Bezier A, Poinssot B, Joubert JM, Pugin A, Heyraud A, Baillieu F (2007) Elicitor and resistance-inducing activities of β-1,4 cellodextrins in grapevine, comparison with β-1,3 glucans and α-1,4 oligogalacturonides. J Exp Bot 58:1463–1472

Bahrini I, Ogawa T, Kobayashi F, Kawahigashi H, Handa H (2011a) Overexpression of the pathogen-inducible wheat *TaWRKY45* gene confers disease resistance to multiple fungi in transgenic wheat plants. Breed Sci 61:319–326

Bahrini I, Sugisawa M, Kikuchi R, Ogawa T, Kawahigashi H, Ban T, Handa H (2011b) Characterization of a wheat transcription factor, *TaWRKY45*, and its effect on *Fusarium* head blight resistance in transgenic wheat plants. Breed Sci 61:121–129

Belbahri L, Boucher C, Candresse T, Nicole M, Ricci P, Keller HA (2001) A local accumulation of the *Ralstonia solanacearum* PopA protein in transgenic tobacco renders a compatible interaction incompatible. Plant J 28:419–430

Bellincampi D, Cervone F, Lionetti V (2014) Plant cell wall dynamics and wall-related susceptibility in plant-pathogen interactions. Front Plant Sci 5:228. doi:10.3389/fpls.2014.00228

Böhm H, Albert I, Fan L, NürnbergerT RA (2014) Immune receptor complexes at the plant surface. Curr Opin Plant Biol 20:47–54

Boller T, He SH (2009) Innate immunity in plants: an arms race between pattern recognition receptors in plants and effectors in microbial pathogens. Science 324:742–744

Borras-Hidalgo O, Caprari C, Hernandez-Estevez I, De Lorenzo G, Cervone F (2012) A gene for plant protection: expression of a bean polygalacturonase inhibitor in tobacco confers a strong resistance against *Rhizoctonia solani* and two oomycetes. Front Plant Sci 3:1–6

Boutrot F, Segonzac C, Chang KN, Qiao H, Ecker JR, Zipfel C (2010) Direct transcriptional control of the *Arabidopsis* immune receptor FLS2 by the ethylene-dependent transcription factors EIN3 and EIL1. Proc Natl Acad Sci U S A 107:14502–14507

Brader G, Djamei A, Teige M, Palva ET, Hirt H (2007) The MAP kinase kinase MKK2 affects disease resistance in *Arabidopsis*. Mol Plant Microbe Interact 20:589–596

Bradley CA (2008) Effect of fungicide seed treatments on stand establishment, seedling disease, and yield of soybean in North Dakota. Plant Dis 92:120–125

Brutus A, Sicilia F, Macone A, Cervone F, De Lorenzo G (2010) A domain swap approach reveals a role of the plant wall-associated kinase 1 (WAK1) as a receptor of oligogalacturonides. Proc Natl Acad Sci U S A 107:9452–9457

Byamukama E, Robertson AE, Nutter FW Jr (2015) *Bean pod mottle virus* time of infection influences soybean yield, yield components, and quality. Plant Dis 99:1026–1032

Cai X-Z, Zhou X, Xu Y-P, Joosten MHAJ, deWit PJGM (2007) *Cladosporium fulvum* CfHNNI1 induces hypersensitive necrosis, defence gene expression and disease resistance in both host and nonhost plants. Plant Mol Biol 64:89–101

Cao Y, Wu Y, Zheng Z, Song F (2006) Overexpression of the rice EREBP-like gene *OsBIERF3* enhances disease resistance and salt tolerance in transgenic tobacco. Physiol Mol Plant Pathol 67:202–211

Cao W-L, Chu R-Z, Zhang Y, Luo J, Su Y-Y, Xie L-J, Zhang H-S, Wang J-F, Bao Y-M (2016) *OsJAMyb*, a R2R3-type MYB transcription factor, enhanced blast resistance in transgenic rice. Physiol Mol Plant Pathol (in press)

Capodicasa C, Vairo D, Zabotina O, McCartney L, Caprari C, Mattei B, Manfredini C, Aracri B, Benen J, Knox JF, De Lorenzo G, Cervone F (2004) Targeted modification of homogalacturonan by transgenic expression of a fungal polygalacturonase alters plant growth. Plant Physiol 135:1294–1304

Century K, Reuber TL, Ratcliffe OJ (2008) Regulating the regulators: the future prospects for transcription-factor-based agricultural biotechnology products. Plant Physiol 147:20–29

Chassot C, Buchala A, Schoonbeek H-J, Metraux J-P, Lamotte O (2008) Wounding of Arabidopsis leaves causes a powerful but transient protection against *Botrytis* infection. Plant J 55:555–567

Chavan V, Kamble A (2013) β-Aminobutyric acid primed expression of WRKY and defence genes in *Brassica carianta* against Alternaria blight. J Phytopathol 161:859–865

Che FS, Nakajima Y, Tanaka N, Iwano M, Yoshida T, Takayama S, Kadota I, Isogai A (2000) Flagellin from an incompatible strain of *Pseudomonas avenae* induces a resistance response in cultured rice cells. J Biol Chem 275:32347–32356

Chen ZY, Brown RL, Rajasekaran K, Damann KE, Cleveland TE (2006) Identification of a maize kernel pathogenesis-related protein and evidence for its involvement in resistance to *Aspergillus flavus* infection and aflatoxin production. Phytopathology 96:87–95

Chen L, Zhang S-J, Zhang S-S, Qu S, Ren X, Long J, Yin Q, Qian J, Sun F, Zhang C, Wang L, Wu X, Wu T, Zhang Z, Cheng Z, Hayes M, Beer SV, Dong H (2008) A fragment of the *Xanthomonas oryzae* pv. *oryzicola* harpin $HpaG_{Xooc}$ reduces disease and increases yield of rice in extensive grower plantings. Phytopathology 98:792–802

Chen F, Gao J, Miao S, Yuan Y-X, Wang M-Y, Li Q, Mao B-Z, Jiang L-W, He ZH (2010a) Plasma membrane localization and potential endocytosis of constitutively expressed XA21 proteins in transgenic rice. Mol Plant 3:917–926

Chen X, Chern M, Canlas PE, Jiang C, Ruan D, Cao P, Ronald PC (2010b) A conserved threonine residue in the juxtamembrane domain of the XA21 pattern recognition receptor is critical for kinase autophosphorylation and XA21-mediated immunity. J Biol Chem 285:10454–10463

Chen X, Zuo S, Schwessinger B, Chern M, Canlas PE, Ruan D, Zhou X, Wang J, Daudi A, Petzold CJ (2014) An XA21-associated kinase (OsSERK2) regulates immunity mediated by the XA21 and XA3 immune receptors. Mol Plant 7:874–892

Cheng B, Yu X, Ma Z, Dong S, Dou D, Wang Y, Zheng X (2012) *Phytophthora sojae* effector Avh331 suppresses the plant defence response by disturbing the MAPK signalling pathway. Physiol Mol Plant Pathol 77:1–9

Cheng H, Liu H, Deng Y, Xiao J, Li X, Wang S (2015) The WRKY45-2 WRKY13 WRKY42 transcriptional regulatory cascade is required for rice resistance to fungal pathogen. Plant Physiol 167:1087–1099

Choi JJ, Klosterman SJ, Hadwiger LA (2004) A promoter from pea gene DRR206 is suitable to regulate an elicitor-coding gene and develop disease resistance. Phytopathology 94:651–660

Choi M-S, Heu S, Paek N-C, Koh H-J, Lee J-S, Oh C-S (2012) Expression of *hpa1* gene encoding a bacterial harpin protein on *Xanthomonas oryzae* pv. *oryzae* enhances disease resistance to both fungal and bacterial pathogens in rice and *Arabidopsis*. Plant Pathol J 28:364–372

Chuang H-W, Chang P-Y, Syu Y (2014) Harpin protein, an elicitor of disease resistance, acts as a growth promoter in *Phalaenopsis* orchids. J Plant Growth Regul 33:788–797. doi:10.1007/s00344-014-9425-1

Chujo T, Takai R, Akimoto-Tomiyama C, Ando S, Minami E, Nagamura Y, Kaku H, Shibuya N, Yasuda M, Nakashita H, Umemura K, Okada K, Nojiri H, Yamane H (2007) Involvement of the elicitor-induced gene OsWRKY53 in the expression of defense-related genes in rice. Biochim Biophys Acta 1769:497–505

Chujo T, Miyamoto K, Shimogawa T, Shimizu T, Otake Y, Yokotani N, Nishizawa Y, Shibuya N, Nojiri H, Yamane H, Minami E, Okada K (2013) OsWRKY28, a PAMP-responsive transrepressor, negatively regulates innate immune responses in rice against blast fungus. Plant Mol Biol 82:23–37

Chujo T, Miyamoto K, Ogawa S, Masuda Y, Shimizu T, Kishi-Kaboshi M, Takahashi A, Nishizawa Y, Minami E, Norjiri H, Yamane H, Okada K (2014) Overexpression of phosphomimic mutated OsWRKY53 leads to enhanced blast resistance in rice. PLoS One 9(6):e98737. doi:10.1371/journal.pone.0098737

Cohen Y, Van den Langenberg KM, Wehner TC, Ojiambo PS, Hausbeck M, Quesada-Ocampo LM, Lebeda A, Sierotzki H, Gisi U (2015) Resurgence of *Pseudoperonospora cubensis*: the causal agent of cucurbit downy mildew. Phytopathology 105:998–1012

Coppinger P, Repetti PP, Day B, Dahlbeck D, Mehlert A, Staskawicz BJ (2004) Overexpression of the plasma membrane-localized NDR1 protein results in enhanced bacterial disease resistance in *Arabidopsis thaliana*. Plant J 40:225–237

Coppola M, Corrado G, Coppola V, Cascone P, Martinelli R, Digilio MC, Pennacchio F, Rao R (2014) Prosystemin overexpression in tomato enhances resistance to different biotic stresses by activating genes of multiple signaling pathways. Plant Mol Biol Rep 33:1270–1285. doi:10.1007/s11105-014-0834-x

Coram TE, Pang ECK (2005) Isolation and analysis of candidate ascochyta blight defence genes in chickpea, Part 1. Generation and analysis of an expressed sequence tag (EST) library. Physiol Mol Plant Pathol 66:192–200

Côté F, Hahn MG (1994) Oligosaccharins: structures and signal transduction. Plant Mol Biol 26:1379–1411

D'Amelio R, Berta G, Gamalero E, Massa N, Avidano L, Cantamessa S, D'Agostino G, Bosco D, Marzachi C (2011) Increased plant tolerance against chrysanthemum yellows phytoplasma (*Candidatus Phytoplasma asteris*) following double inoculation with *Glomus mosseae* BEG12 and *Pseudomonas putida* SIPflRif. Plant Pathol 60:1014–1022

Dafermos NG, Kasselaki AM, Goumas DE, Spantidakis K, Eyre MD, Leifert C (2012) Integration of elicitors and less-susceptible hybrids for the control of powdery mildew in organic tomato crops. Plant Dis 96:1506–1512

Dai S, Wei X, Alfonso AA, Pei L, Duque UG, Zhang Z, Babb GM, Beachy RN (2008) Transgenic rice plants that overexpress transcription factors RF2a and RF2b are tolerant to rice tungro virus replication and disease. Proc Natl Acad Sci U S A 105:21012–21016

Dang F, Wang Y, She J, Lei Y, Liu Z, Eulgem T, Lai Y, Lin J, Yu L, Lei D, Guan D, Li X, Yuan Q, He S (2014) Overexpression of *CaWRKY27*, a subgroup IIe transcription factor of *Capsicum annuum*, positively regulates tobacco resistance to *Ralstonia solanacearum* infection. Physiol Plant 150:397–411

de Capdeville G, Beer SV, Wilson CL, Aist JR (2002) Alternative disease control agents induced resistance to blue mold in harvested 'Red Delicious' apple fruit. Phytopathology 92:900–908

de Capdeville G, Beer SV, Watkins CB, Wilson CL, Tedeschi LO, Aist JR (2003) Pre- and post-harvest harpin treatments of apples induce resistance to blue mold. Plant Dis 87:39–44

de Capdeville GD, Beer SV, Wilson CL, Aist JR (2008) Some cellular correlates of harpin-induced resistance to blue mold of apples. Trop Plant Pathol 33:103–113

de Jonge R, van Esse HP, Kombrink A, Shinya T, Desaki Y, Bours R, van der Krol S, Shibuya N, Joosten MHAJ, Thomma BPHJ (2010) Conserved fungal LysM effector Ecp6 prevents chitin-triggered immunity in plants. Science 329:953–955

De Lorenzo G, Brutus A, Savatin DY, Sicilia F, Cervone F (2011) Engineering plant resistance by constructing chimeric receptors that recognize damage-associated molecular patterns (DAMPs). FEBS Lett 585:1521–1526

Denoux C, Galletti R, Mammarella N, Gopalan S, Werck D, De Lorenzo G, Ferrari S, Ausubel FM, Dewdney J (2008) Activation of defense response pathways by OGs and Flg22 elicitors in Arabidopsis seedlings. Mol Plant 1:423–445

Di Matteo A, Giovane A, Raiola A, Camardella L, Bonivento D, De Lorenzo G, Cervone F, Bellincampi D, Tsernoglou D (2005) Structural basis for the interaction between pectin methylesterase and a specific inhibitor protein. Plant Cell 17:849–858

Dodds PN, Rathjen JP (2010) Plant immunity: towards an integrated view of plant-pathogen interactions. Nat Rev Genet 11:539–548

Dong H-P, Peng J, Bao Z, Meng X, Bonasera JM, Chen G, Beer SV, Dong H (2004) Downstream divergence of the ethylene signaling pathway for harpin-stimulated Arabidopsis growth and insect resistance. Plant Physiol 136:3628–3638

Donghua J, Zejian G, Xujun C, Zhiqiang C, Zhong Z (2004) Study on tobacco disease resistances mediated by the elicitor gene cryptogein from *Phytophthora cryptogea*. Chin J Agric Biotechnol 1:125–131

Elmer PAG, Reglinski T (2006) Biosuppression of *Botrytis cinerea* in grapes. Plant Pathol 55:155–177

Fan H, Wang F, Gao H, Wang L, Xu J, Zhao Z (2011) Pathogen-induced *MdWRKY1* in 'Qinguan' apple enhances disease resistance. J Plant Biol 54:150–158

Farag MA, Huhman DV, Dixon RA, Sumner LW (2008) Metabolomics reveals novel pathways and differential mechanistic and elicitor-specific responses in phenylpropanoid and isoflavonoid biosynthesis in *Medicago truncatula* cell cultures. Plant Physiol 1486:387–402

Federici L, Di Matteo A, Fernandez-Recio J, Tsernoglou D, Cervone F (2006) Polygalacturonase-inhibiting proteins: players in plant immunity? Trends Plant Sci 11(2):65–70

Fernández-Ortuño D, Grabke A, Li X, Schnabel G (2015) Independent emergence of resistance to seven chemical classes of fungicides in *Botrytis cinerea*. Phytopathology 105:424–432

Ferrari S, Galletti R, Denoux C, De Lorenzo G, Ausubel FM, Dewdney J (2007) Resistance to *Botrytis cinerea* induced in Arabidopsis by elicitors is independent of salicylic acid, ethylene, or jasmonate signaling but requires PHYTOALEXIN DEFICIENT3. Plant Physiol 144:367–379

Ferrari S, Galletti R, Pontiggia D, Manfredni D, Lionetti V, Bellincampi D, Cervone F, De Lorenzo G (2008) Transgenic expression of a fungal *endo*-polygalacturonase increases plant resistance to pathogens and reduces auxin sensitivity. Plant Physiol 146:669–681

Ferrari S, Sella L, Janni M, De Lorenzo G, Favaron F, D'Ovidio R (2012) Transgenic expression of polygalacturonase-inhibitimg proteins in *Arabidopsis* and wheat increases resistance to the flower pathogen *Fusarium graminearum*. Plant Biol 14:31–38

Ferrari S, Savatin DV, Sicilia F, Gramegna G, Cervone F, Lorenzo GD (2013) Oligogalacturonides: plant damage-associated molecular patterns and regulators of geowth and development. Front Plant Sci 4:49. doi:10.3389/fpls.2013.00049

Fischer U, Dröge-Laser W (2004) Overexpression of *Nt*ERF5, a new member of the tobacco ethylene response transcription factor family enhances resistance to *Tobacco mosaic virus*. Mol Plant Microbe Interact 17:1162–1171

Flors V, Ton J, van Doorn R, Garcia-Agustin P, Mauch-Mani B (2008) Interplay between JA, SA, and ABA signaling during basal and induced resistance against *Pseudomonas syringae* and *Alternaria brassicicola*. Plant J 54:81–92

Fu M, Xu M, Zhou T, Wang D, Tian S, Han L, Dong H, Zhang C (2014) Transgenic expression of a functional fragment of harpin protein Hpa1 in wheat induces the phloem-based defence against English grain aphid. J Exp Bot 65:1439–1453

Fujiwara S, Tanaka N, Kaneda T, Takayama S, Isogai A, Che F-S (2004) Rice cDNA microarray-based gene expression profiling of the response to flagellin perception in cultured rice cells. Mol Plant Microbe Interact 17:986–998

Fung RWM, Gonzalo M, Fekete C, Kovacs LG, He Y, Marsh E, McIntyre LM, Schchtman DP, Qiu W (2008) Powdery mildew induces defense-oriented reprogramming of the transcriptome in a susceptible but not in a resistant grapevine. Plant Physiol 146:236–249

Gent DH, Twomey MC, Wolfenbarger SN, Woods JL (2015) Pre- and postinfection activity of fungicides in control of hop downy mildew. Plant Dis 99:858–865

Gimenez-Ibanez S, Rathjen JP (2010) The case for the defense: plants versus *Pseudomonas syringae*. Microbes Infect 12:428–437

Gimenez-Ibanez S, Hann DR, Ntoukakis V, Petutschnig E, Lipka V, Rathjen JP (2009a) AvrPtoB targets the LysM receptor kinase CERK1 to promote bacterial virulence on plants. Curr Biol 19:423–429

Gimenez-Ibanez S, Ntoukakis V, Rathjen JP (2009b) The LysM receptor kinase CERK1 mediates bacterial perception in Arabidopsis. Plant Signal Behav 4:539–541

Giovane A, Servillo L, Balestrieri C, Raiola A, D'Arvino R, Tamburrini M, Ciardiello MA, Camardella L (2004) Pectin methylesterase inhibitor. Biochim Biophys Acta 1696:245–252

Göhre V, Spallek T, Häweker H, Mersmann S, Mentzel T, Boller T, de Torres M, Mansfield JW, Robatzek S (2008) Plant pattern-recognition receptor FLS2 is directed for degradation by the bacterial ubiquitin ligase AvrPtoB. Curr Biol 18:1824–1832

Goto S, Sasakura-Shimoda F, Suetsugu M, Selvaraj MG, Hayashi N, Yamazaki M, Ishitani M, Shimono M, Sugano S, Matsushita A, Tanabata T, Takatsuji H (2015) Development of disease-resistant rice by optimized expression of WRKY45. Plant Biotechnol J 13:753–765

Groβkinsky DK, Koffler BE, Roitsch T, Maier R, Zechmann B (2012) Compartment-specific antioxidative defense in *Arabidopsis* against virulent and avirulent *Pseudomonas syringae*. Phytopathology 102:662–673

Gudmestad NC, Arabiat S, Miller JS, Pasche JS (2013) Prevalence and impact of SDHI fungicide resistance in *Alternaria solani*. Plant Dis 97:952–960

Guo Z, Chen XJ, Wu XL, Ling JQ, Xu P (2004) Overexpression of the AP2/EREBP transcription factor OPBP1 enhances disease resistance and salt tolerance in tobacco. Plant Mol Biol 55:607–618

Gust AA, Brunner F, Nürnberger T (2010) Biotechnological concepts for improving plant innate immunity. Curr Opin Biotechnol 21:204–210

Hamiduzzaman MM, Jakeb G, Barnavon L, Neuhaus JM, Mauch-Mani B (2005) β-Aminobutyric acid-induced resistance against downy mildew in grapevine acts through the potentiation of callose formation and jasmonic acid signaling. Mol Plant Microbe Interact 18:819–829

Han M, Ryu H-S, Kim C-Y, Park D-S, Ahn Y-K, Jeon J-S (2013) *OsWRKY30* is a transcription activator that enhances rice resistance to the *Xanthomonas oryzae* pathovar oryzae. J Plant Biol 56:258–265

Han DJ, Wang QL, Chen XM, Zeng QD, Wu JH, Xue WB, Zhan GM, Huang LL, Kang ZS (2015) Emerging Yr26-virulent races of *Puccinia striiformis* f. *tritici* are threatening wheat production in the Sichuan Basin, China. Plant Dis 99:754–760

Handiseni M, Jo Y-K, Zhou X-G (2015) Integration of *Brassica* cover crop with host resistance and azoxystrobin for management of rice sheath blight. Plant Dis 99:883–885

Hann DR, Rathjen JP (2007) Early events in the pathogenecity of *Pseudomonas syringae* on *Nicotiana benthamiana*. Plant J 49:607–618

Hann DR, Gimenez-Ibanez S, Rathjen JP (2010) Bacterial virulence effectors and their activities. Curr Opin Plant Biol 13:388–393

Hann CT, Bequette CJ, Dombrowski JE, Stratmann JVV (2014) Methanol and ethanol modulate responses to danger- and microbe-associated molecular patterns. Front Plant Sci 5:550. doi:10.3389/fpls.2014.00550

He P, Warren RF, Zhao TH, Shan LB, Zhu LH, Tang XY, Zhou JM (2001) Overexpression of Pti5 in tomato potentiates pathogen-induced defense gene expression and enhances disease resistance to *Pseudomonas syringae* pv. *tomato*. Mol Plant Microbe Interact 14:1453–1457

Hermosa MR, Turra D, Fogliano V, Monte E, Lorito M (2006) Identification and characterization of potato protease inhibitors able to inhibit pathogenicity and growth of *Botrytis cinerea*. Physiol Mol Plant Pathol 68:138–148

Hernández-Blanco C, Feng DX, Hu J, Sanchez-Vallet A, Deslandes L, Llorente F, Berrocal-Lobo M, Keller H, Barlet X, Sánchez-Rodriguez C, Anderson LK, Somerville S, Marco Y, Molina A (2007) Impairment of cellulose synthase required for *Arabidopsis* secondary cell wall formation enhances disease resistance. Plant Cell 19:890–903

Higashi K, Ishiga Y, Inagaki Y, Toyoda K, Shiraishi T, Ichinose Y (2008) Modulation of defense signal transduction by flagellin-induced WRKY41 transcription factor in *Arabidopsis thaliana*. Mol Genet Genomics 279:303–312

Holmes GJ, Ojiambo PS, Hausbeck MK, Quesada-Ocampo L, Keinath AP (2015) Resurgence of cucurbit downy mildew in the United States: a watershed event for research and extension. Plant Dis 99:428–441

Hou S, Wang X, Chen D, Yang X, Wang M, Turrà D, Di Pietro A (2014) The secreted peptide PIP1 amplifies immunity through receptor-like kinase 7. PLoS Pathog 10(9):e1004331. doi:10.1371/journal.ppat.1004331

Hu M-J, Grabke A, Dowling ME, Holstein HJ, Schnabel G (2015) Resistance in *Colletotrichum siamense* from peach and blueberry to thiophanate-methyl and azoxystrobin. Plant Dis 99:806–814

Huffaker A, Ryan CA (2007) Endogenous peptide defense signals in *Arabidopsis* differentially amplify signaling for the innate immune response. Proc Natl Acad Sci U S A 104:10732–10736

Huffaker A, Pearce G, Ryan CA (2006) An endogenous peptide signal in *Arabidopsis* activates components of the innate immune response. Proc Natl Acad Sci U S A 103:10098–10103

Huffaker A, Dafoe NJ, Schmelz EA (2011) ZmPep1, an ortholog of Arabidopsis elicitor peptide 1, regulates maize innate immunity and enhances disease resistance. Plant Physiol 155:1325–1338

Huo R, Wang Y, Ma L, Qiao J, Shao M, Gao X (2010) Assessment of inheritance pattern and agronomic performance of transgenic rapeseed having harpinXooC-encoding *hrf2* gene. Transgenic Res 19:841–847

Hwang IS, Hwang BK (2011) The pepper mannose-binding lectin gene *CaMBL1* is required to regulate cell death and defense responses to microbial pathogens. Plant Physiol 155:447–463

Iriti M, Vitalini S, Di Tommaso G, D'Amico S, Borgo M, Faoro F (2011) New chitosan formulation prevents grapevine powdery mildew infection and improves polyphenol content and free radical scavenging activity of grape and wine. Aust J Grape Wine Res 17:263–269

Jakab G, Cottier V, Toquin V, Rigoli G, Zimmerli L, Metraux JP, Mauch-Mani B (2001) β-aminobutyric acid-induced resistance in plants. Eur J Plant Pathol 107:29–37

Jang YS, Sohn SI, Wang MH (2006) The *hrpN* gene of *Erwinia amylovora* stimulates tobacco growth and enhances resistance to *Botrytis cinerea*. Planta 223:449–456

Jeworutzki E, Roelfsema MR, Anschütz U, Krol E, Elzenga JT, Felix G, Boller T, Hedrich R, Becker D (2010) Early signaling through the *Arabidopsis* pattern recognition receptors FLS2 and EFR involves Ca^{2+}-associated opening of plasma membrane anion channels. Plant J 62:367–378

Jisha V, Dampanaboina L, Vadassery J, Mithofer A, Kappara S, Ramanan R (2015) Overexpression of an AP2/ERF type transcription factor *OsEREBP1* confers biotic and abiotic stress tolerance in rice. PLoS One 10(6):e0127831. doi:10.1371/journal.pone.0127831

Jones AL (2001) Challenges of controlling fire blight. Compact Fruit Tree 34(3):86–91

Joubert DA, Slaughter AR, Kemp G, Becker JV, Krooshof GH, Bergmann C, Benen J, Pretorius IS, Vivier MA (2006) The grapevine polygalacturonase-inhibiting protein (VvPGIP1) reduces *Botrytis cinerea* susceptibility in transgenic tobacco and differentially inhibits fungal polygalacturonases. Transgenic Res 15:702–887

Joubert DA, Kars I, Wagemakers L, Bergmann C, Kemp G, Vivier MA, van Kan JA (2007) A polygalacturonase-inhibiting protein from grapevine reduces the symptoms of the endopolygalacturonase BcPG2 from *Botrytis cinerea* in *Nicotiana benthamiana* leaves without any evidence for in vitro interaction. Mol Plant Microbe Interact 20:392–402

Kanetis L, Förster H, Adaskaveg JE (2008) Optimizing efficacy of new postharvest fungicides and evaluation of sanitizing agents for managing citrus green mold. Plant Dis 92:261–269

Keeling CI, Weisshaar S, Lin RPC, Bohlmann J (2008) Functional plasticity of paralogous diterpene synthases involved in conifer defense. Proc Natl Acad Sci U S A 105:1090–1985

Keinath AP (2015) Baseline sensitivity of *Didymella bryoniae* to cyprodinil and fludioxonil and field efficacy of these fungicides against isolates resistant to pyraclostrobin and boscalid. Plant Dis 99:836–841

Keller H, Pamboukdjian N, Ponchet M, Poupet A, Delon R, Verrier J-L, Roby D, Ricci P (1999) Pathogen-induced elicitin production in transgenic tobacco generates a hypersensitive response and nonspecific disease resistance. Plant Cell 11:223–236

Kim S-Y, Kim Y-C, Seong ES, Lee Y-H, Park M, Choi D (2007) The chili pepper CaATL1: an AT-hook motif-containing transcription factor implicated in defence responses against pathogens. Mol Plant Pathol 8:761–771

Kitner M, Lebeda A, Sharma R, Runge F, Dvořák P, Tahir A, Choi Y-J, Sedláková B, Thines M (2015) Coincidence of virulence shifts and population genetic changes of *Pseudoperonospora cubensis* in the Czech Republic. Plant Pathol 64:1461–1470

Knecht K, Seyffarth M, Desel C, Thurau T, Sherameti I, Lou B, Oelmüller R, Cai D (2010) Expression of *BvGLP-1* encoding a germin-like protein from sugar beet in *Arabidopsis thaliana* leads to resistance against phytopathogenic fungi. Mol Plant Microbe Interact 23:446–457

Krol E, Mentzel T, Chinchilla D, Boller T, Felix G, Kemmerling B, Postel S, Arents M, Jeworutzki E, Al-Rasheid KAS, Becker D, Hedrich R (2010) Perception of the Arabidopsis danger signal